Kidney International (2007) **71**, 181. doi:10.1038/sj.ki.5002095

Severe protein restriction reduces blood pressure

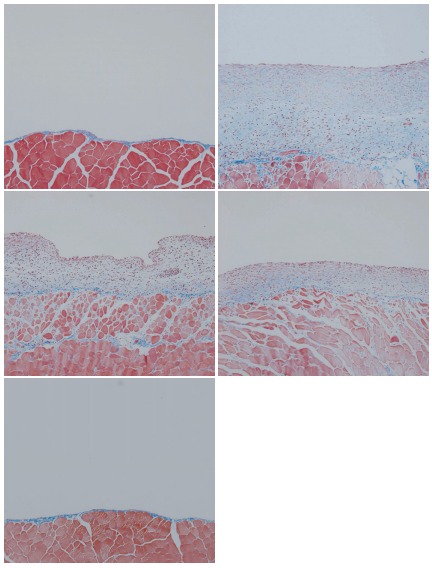
Hypertension is a well-known concomitant of chronic kidney disease and can clearly be controlled by removal of fluid during dialysis. Bellizzi *et al.* in this issue studied the effect of dietary management on blood pressure control in severe renal disease. They placed patients on three diets of varying protein restriction. Some were placed on a low-protein diet, others were placed on severe protein restriction supplemented by the ketoanalogs of essential amino acids, and a third group were placed on an *ad libitum* diet. After the patients were placed on these diets, their dietary protein intake 6 months later reflected the initial prescription. The blood pressure decreased only in patients on the very-low-protein diet despite reduction of antihypertensive drugs. Urinary urea excretion directly correlated with urinary sodium excretion, which diminished in the patients on the very-low-protein diet. Daily sodium excretion was also measured. The authors found that the blood pressure was independently related to urinary sodium excretion and the very-low-protein diet, but not to the level of protein intake. The results demonstrate the primacy of salt excretion, that is, salt intake in the hypertension of chronic kidney disease, but also that severe protein restriction (while maintaining essential amino acid intake) might also play a part. **See page 245.**

Obesity, hyperfiltration, and salt

There is no doubt that obesity increases the risk of long-term renal damage. In addition, there have been syndromes of specific glomerulosclerosis that seems to be associated with massive obesity. What could be the mechanism for this? Krikken *et al.* in this issue propose that renal hemodynamics in obese people might be involved in this problem; they studied the interaction between a high-salt diet and renal blood flow and glomerular filtration rate. They placed healthy subjects on low- or high-salt diets and found that the filtration rate and the effective renal plasma flow increased when subjects were changed from a low-salt to a high-salt diet, as has been found previously. They found that there was a good correlation between the glomerular filtration rate or filtration fraction and the body mass index, but only during high-salt feeding. Interestingly, for subjects with a high body mass index, the effects of a high-salt diet on the filtration rate and filtration fraction were significantly different. The filtration fraction was significantly higher in obese subjects, but only during a high-salt diet. These results suggest that high-salt feeding in obese subjects causes hyperfiltration and may contribute to the kidney damage seen in obesity. One should point out that the high-salt diet contained 200 mmol of Na, close to the average diet of industrialized countries. **See page 260.**

Endostatin in peritoneal fibrosis

Peritoneal fibrosis is one of the major complications of chronic ambulatory peritoneal dialysis. Studies in experimental and human dialysis showed that new vessel formation seems to be involved in the fibrosis, and it seems that vascular endothelial growth factor (VEGF) is increased under some circumstances of peritoneal dialysis. In this issue, Tanabe *et al.* examine the efficacy of endostatin in preventing peritoneal fibrosis. Endostatin is a



peptide fragment from collagen XI that is known to be antiangiogenic in a variety of systems. The authors induced peritoneal inflammation by instilling chlorhexidine gluconate into the peritoneal cavities of mice and found that subcutaneous delivery of endostatin reduced the accumulation of collagen I and collagen III. As expected from its antiangiogenic action, it also reduced the number of blood vessels. There was also a reduction in macrophages. Endostatin also reduced the expression of VEGF-A and TGF-β1. There was a reduction in cells expressing α-smooth muscle actin. All of these results suggest that endostatin might be a useful reagent to reduce inflammation in the peritoneum. There is much interest in the use of endostatin for malignant tumors, and the results of Tanabe *et al.* suggest that, at least in this system of peritoneal inflammation, endostatin might be a useful adjunct in the fibrosis seen during peritoneal dialysis. However, it must be said that the model was one where inflammation was produced not through recurrent dialyses but rather through the injection of an inflammatory agent. Regardless, the results clearly have therapeutic potential. **See page 227.**